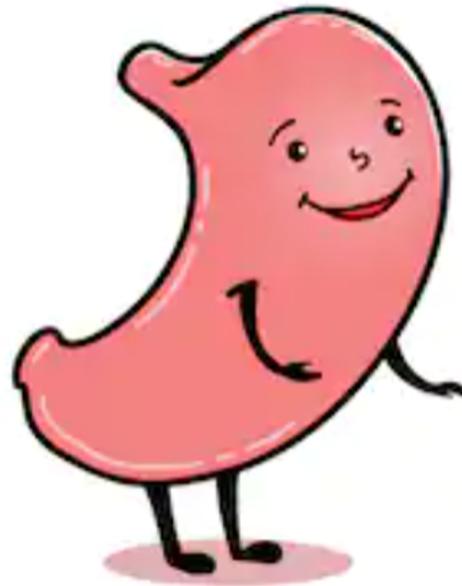


DAVID 'GORD'TON

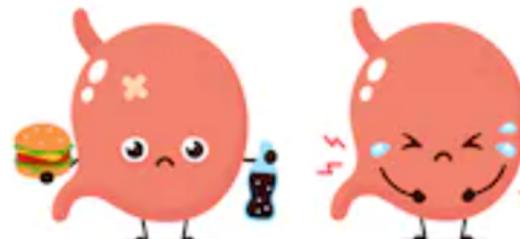
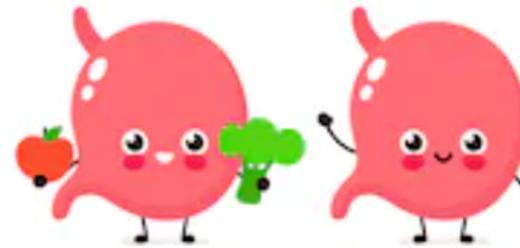
By Chloe Nettleton and
Freddie Weyman

WELCOME TO LIFE MAINTENANCE!



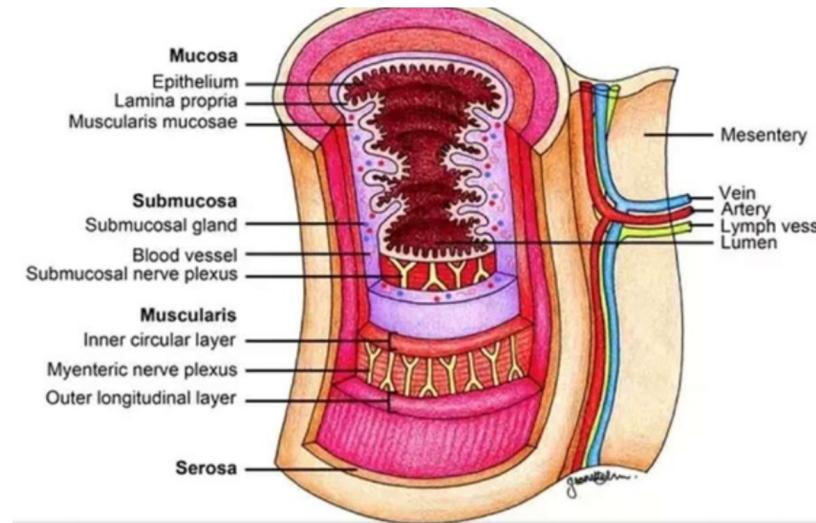
SESSION PLAN

- Anatomy of the upper GI tract
- Stomach physiology
- GORD
- Peptic ulcers
- DDx of abdominal pain

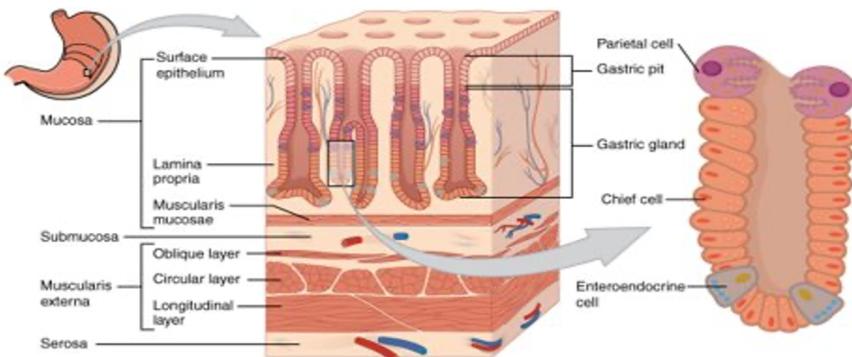
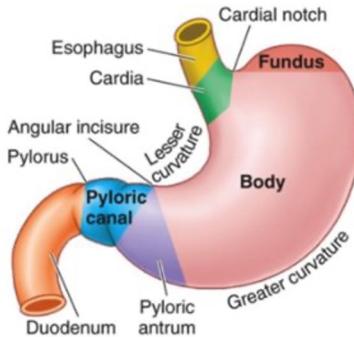


ANATOMY OF THE UPPER GI TRACT

- The oesophagus is a retroperitoneal fibromuscular tube which transports food from the pharynx to the stomach- from C6 to T11
- There are 2 oesophageal sphincters:
 - Upper- at the junction between the pharynx and oesophagus-anatomical as produced by cricopharyngeus
 - Lower- at the junction to the stomach, physiological due to the angle, pressure and mucosal folds
- Histological layers:
 - Adventitia- outer connective tissue
 - Muscularis- outer longitudinal and inner circular, plexus
 - Submucosa- plexus
 - Mucosa- non keratinised stratified squamous epithelium



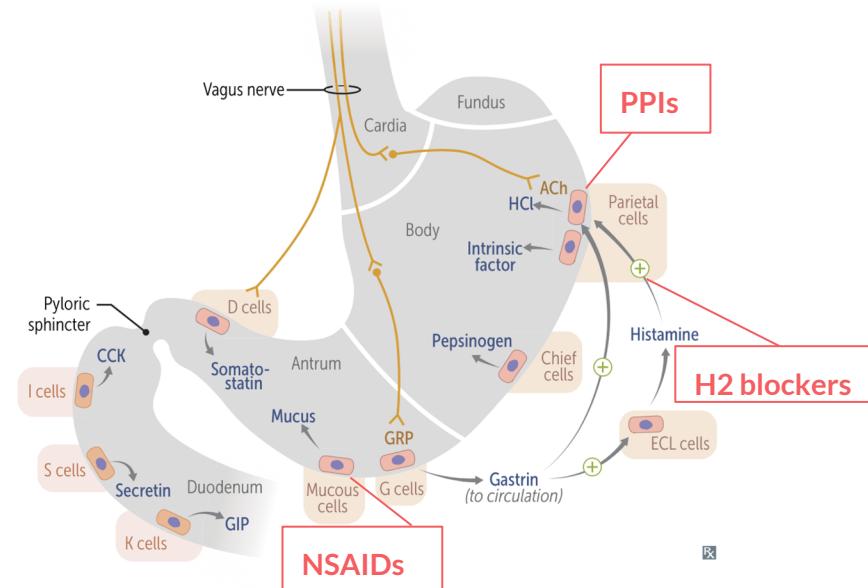
ANATOMY OF THE UPPER GI TRACT



- The stomach is intraperitoneal and lies within the epigastric region of the abdomen
 - There are 4 main divisions (cardia, fundus, body and pylorus) and a greater and lesser curvature
 - The stomach has 2 sphincters:
 - The inferior oesophageal sphincter- allows food to pass into the stomach, involuntary control
 - Pyloric sphincter- controls the exit of chyme
- Histological layers:
- Serosa- connective tissue
 - Muscularis- outer longitudinal, middle circular and inner oblique, plexus
 - Submucosa- plexus
 - Mucosa- simple columnar epithelium and cells for digestion

HOW THE STOMACH SECRETES ACID

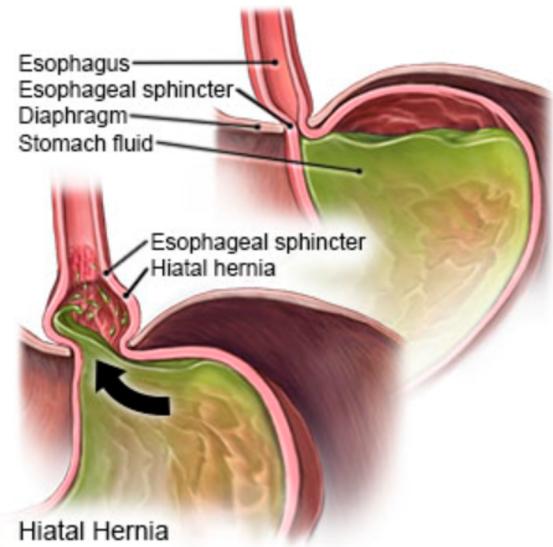
CELL	SECRETION	RECEPTORS	ACTION
Mucus secreting cells	Mucus	• Prostaglandin receptor	Protect the stomach from bacterial infection and acidic environment
Chief cells	Pepsinogen		Protein digestion
G cells	Gastrin		Secretes gastrin into the bloodstream, which binds to enterochromaffin cell to release histamine/parietal cell Promotes HCl production
Enterochromaffin cells	Histamine	• Gastrin receptor • Muscarinic receptor	Histamine binds to H2 receptor on parietal cells to promote HCl production
Parietal cells	HCl	• H2 receptor • Gastrin receptor • Muscarinic receptor • Prostaglandin receptor	Work via a H+/K+ proton pump and a K+/Cl- proton pump to secrete stomach acid
D cells	Somatostatin		Inhibits enterochromaffin/parietal/G cells from producing HCl
COX enzyme (not a cell!)	PGE2 PGI2		Inhibits HCl production via inhibition of parietal cells and promotion of mucus production



Gastrin ↑ acid secretion primarily through its effects on enterochromaffin-like (ECL) cells (leading to histamine release) rather than through its direct effect on parietal cells.

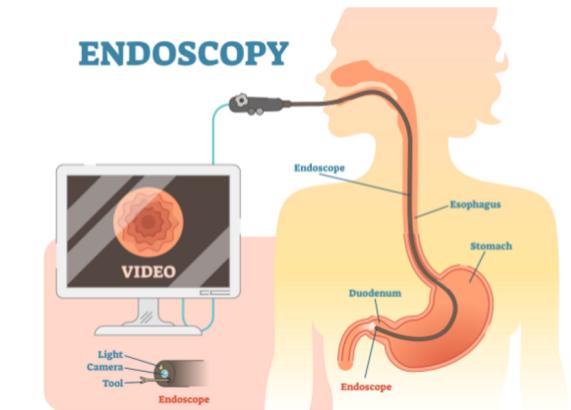
GORD

- **Definition:** A chronic condition in which retrograde flow of stomach contents into the oesophagus causes irritation to the epithelial lining
 - Primarily caused by inappropriate, transient relaxation of the lower oesophageal sphincter
 - Gastroesophageal reflux can occur in healthy individuals after greasy food/alcohol but when it causes troublesome symptoms or oesophageal injury, this is gastroesophageal reflux disease
- **Risk factors:**
 - Family history of GORD
 - Older age
 - Hiatus hernia
 - Obesity
 - Lifestyle habits- smoking, caffeine, alcohol consumption (all decrease LOS tone)
 - Dietary habits- caffeine, carbonated drinks, chocolate, citrus, spicy food
 - Stress
 - Pregnancy (GORD is present in 80% of pregnancies)



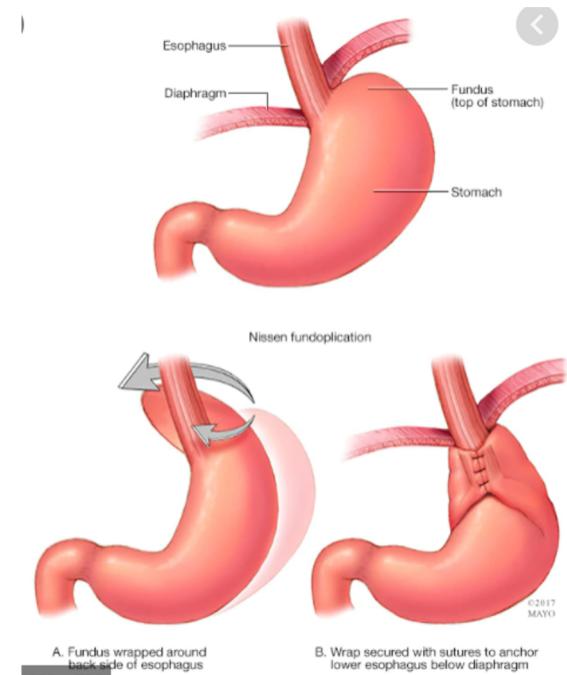
GORD- Presentation

TYPICAL SYMPTOMS	ALARM/ATYPICAL SYMPTOMS
<ul style="list-style-type: none">• Heartburn- a retrosternal burning pain<ul style="list-style-type: none">◦ Worse when lying down or at night• Belching or regurgitation• Pressure sensation in chest• Chronic non-productive cough/nocturnal cough• Halitosis <p>-> clinical diagnosis, trial on PPI</p>	<ul style="list-style-type: none">• Weight loss• Dysphagia• Anaemia• Upper GI bleeding• Persistent vomiting• Odynophagia• Haematemesis and melaena• Abdominal mass• Raised platelet count

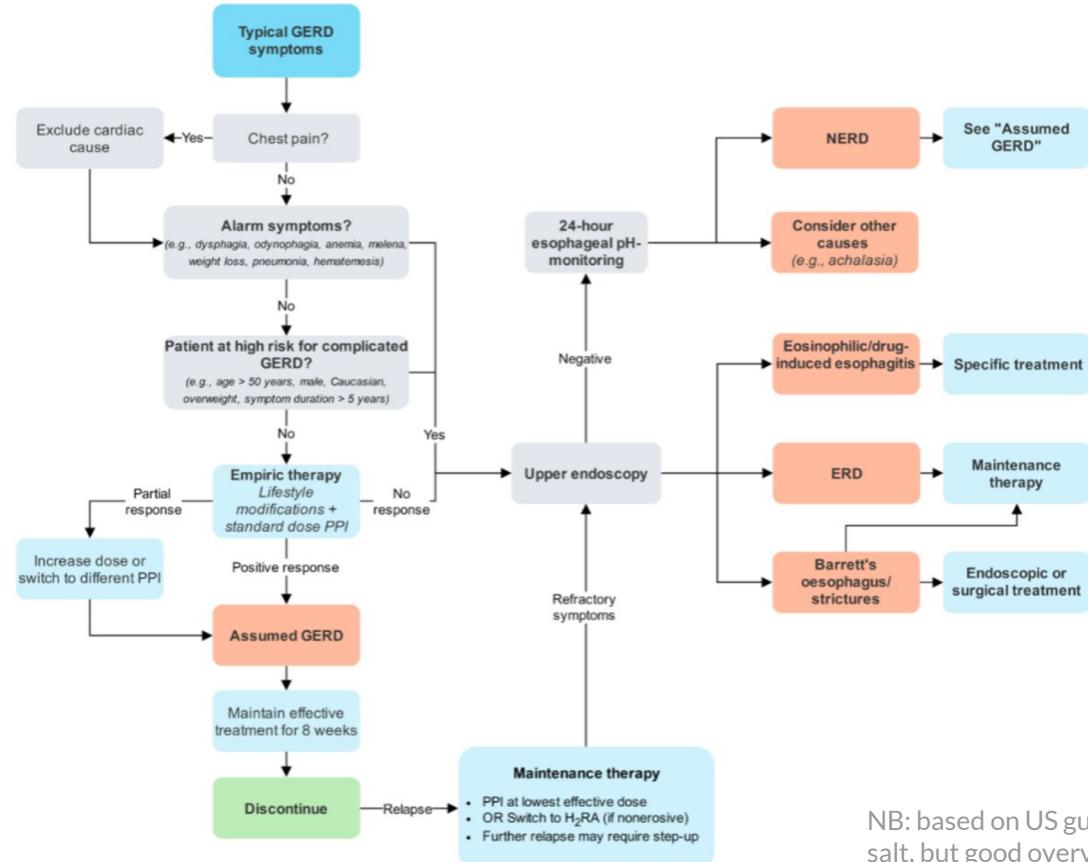


GORD- Management

- Conservative:
 - Diet- small portions, avoid eating <3 hours before bedtime, avoid high fat foods
 - Physical- reduce body weight, elevate the head at night
 - Avoid toxins- nicotine, coffee, alcohol and certain drugs (calcium channel blockers/diazepam)
- Medical:
 - 1st line- standard dose of PPI for at least 4 weeks
 - If no response- need further investigations
 - Partial response- increase the dose/switch PPI
 - Good response- discontinue PPI after 4 weeks
 - Maintenance therapy- if symptoms recur after discontinuation of PPI/complications
 - After 4 weeks, reduce PPI to lowest effective dose or switch to H2RAs
- Surgical:
 - Fundoplication- gastric fundus is wrapped around the LES to form a cuff



GORD- Summary of Management



NB: based on US guidelines so take with a pinch of salt, but good overview of general principles!

HISTOLOGICAL TERMS

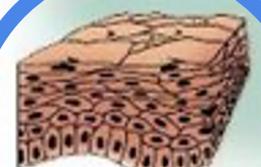
Types of Epithelium



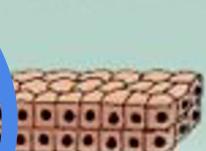
Simple squamous



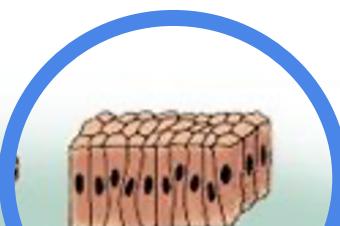
Simple cuboidal



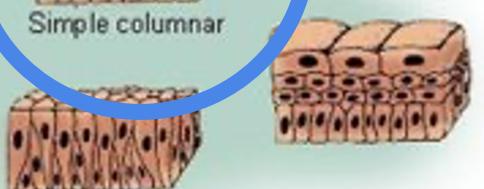
Stratified squamous



Stratified cuboidal

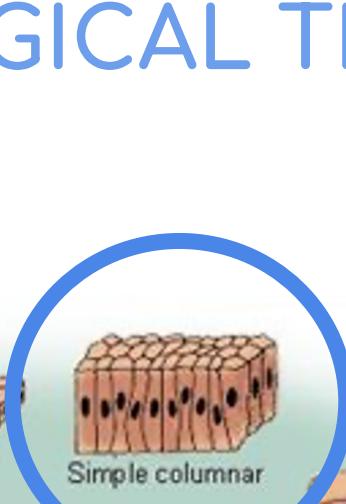


Simple columnar

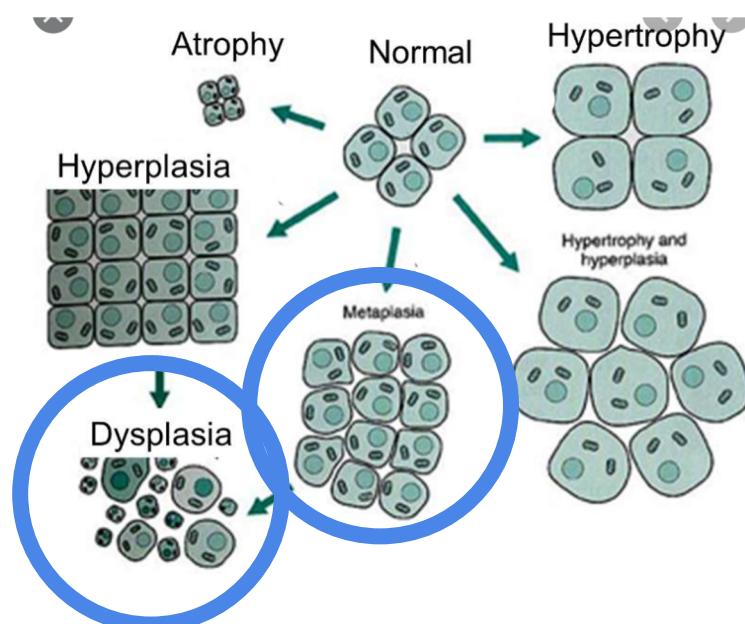


Pseudostratified columnar

Transitional



Transitional



IRON DEFICIENCY ANAEMIA

- Mucosal erosions and ulcerations leads to chronic bleeding

OESOPHAGEAL STRICTURE

- Due to reflux oesophagitis healing with fibrosis
- Causes solid food dysphagia

ASPIRATION OF GASTRIC CONTENTS

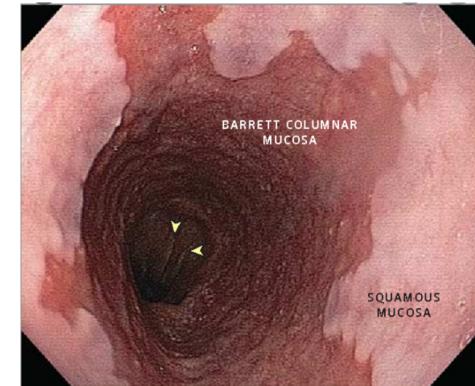
- Aspiration pneumonia
- Chronic bronchitis
- Asthma exacerbations

GORD Complications



BARRETT'S OESOPHAGUS

- Stomach acid damages oesophageal squamous epithelium, undergo metaplasia to be replaced by columnar epithelium and Goblet cells
- A precancerous condition for adenocarcinoma
- Management:
 - Medically with PPIs
 - Endoscopy for to check for dysplasia



PEPTIC ULCERS

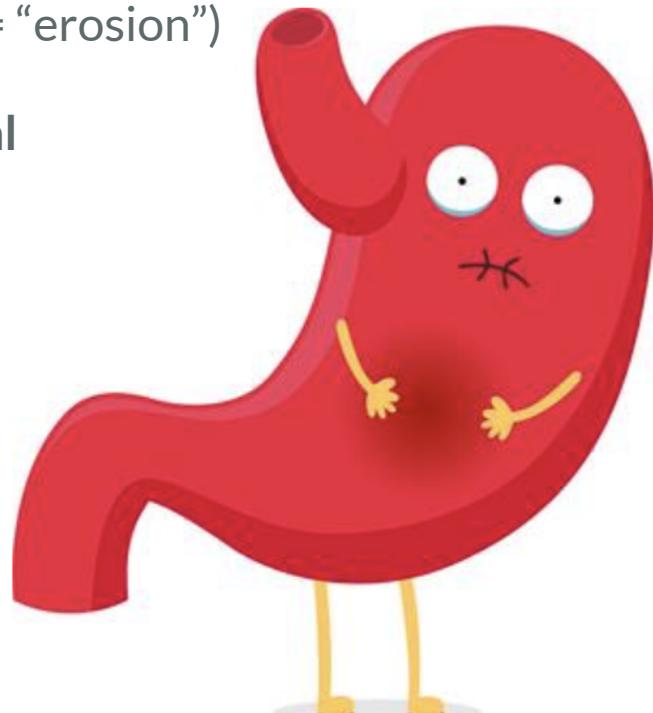
Definition: A break in the mucosal lining of the stomach or duodenum more than 5 mm in diameter, with depth to the submucosa. (Smaller = “erosion”)

Peptic is the umbrella term for either **gastric** or **duodenal**

↑Acid and other damaging factors

and/or

↓Mucus and other protective factors



PEPTIC ULCERS- Presentation

Gastric

Less common than duodenal
60-90% caused by *H. pylori* infection; rest tends to be NSAIDs
Pain often worse with eating, rarely at night
Weight loss
Vomiting more common
May progress to cancer (so always needs biopsy)
Usually on lesser curvature of the stomach

Duodenal

2-3x as common as gastric
>90% caused by *H. pylori* infection
Pain may be relieved by eating, worse several hours after or at night
Weight gain
Vomiting less common
Rarely progress to cancer
Usually in D1 (anterior more common but posterior more deadly)

Epigastric pain
Nausea
Melaena if severe (tarry black stools)
Anaemia if severe (tachycardic, SOB, pallor)
Associated with smoking and increased age



Helicobacter pylori thought to

1. Increase gastrin secretion by locally reducing pH
2. Impair mucus production
3. Cause inflammation

PEPTIC ULCERS- Investigations

Anyone with dysphagia OR over 55y with weight loss and upper abdominal pain/reflux/dyspepsia: **definitely upper endoscopy within 2 weeks**

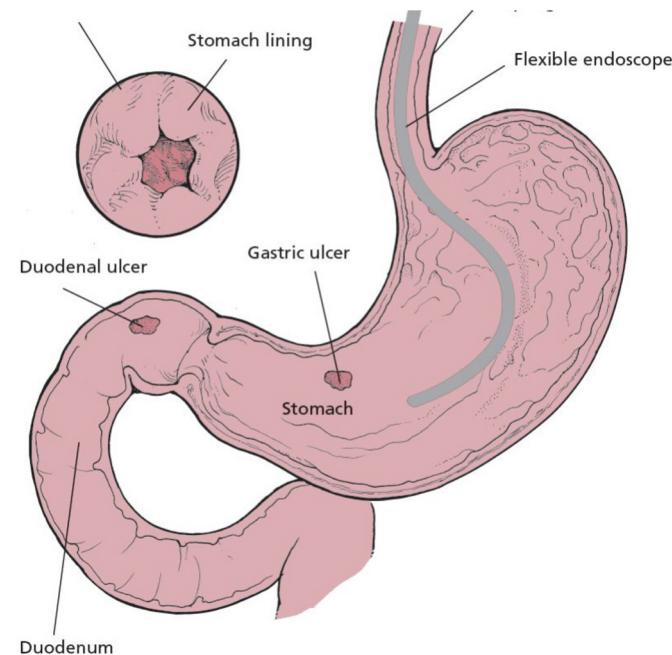
Anyone with haematemesis OR

Over 55y with upper abdominal pain and anaemia

Over 55y with raised platelets and any of: nausea vomiting, weight loss, reflux, dyspepsia, upper abdominal pain

Over 55y with nausea/vomiting and any of: weight loss, reflux, dyspepsia, upper abdominal pain: **consider upper endoscopy within 2 weeks**

Everyone else: **“test and treat” for H. pylori** (stool antigen test cheapest/most common, or carbon-13 urea breath test – no PPIs 2 weeks before, no antibiotics 4 weeks before)



PEPTIC ULCERS- Management

1. If *H. pylori* positive, 7 days of triple therapy (PPI + amoxicillin + clarithromycin/metronidazole) and retest for *H. pylori* after 6-8w
1. If *H. pylori* negative, try 4-8w weeks of PPI or H2 blocker, and make sure NSAIDs are stopped
1. If refractory, endoscopy if not already done, exclude malignancy e.g. gastrin-secreting tumour, other ulcer-causing medicines like steroids and SSRI antidepressants



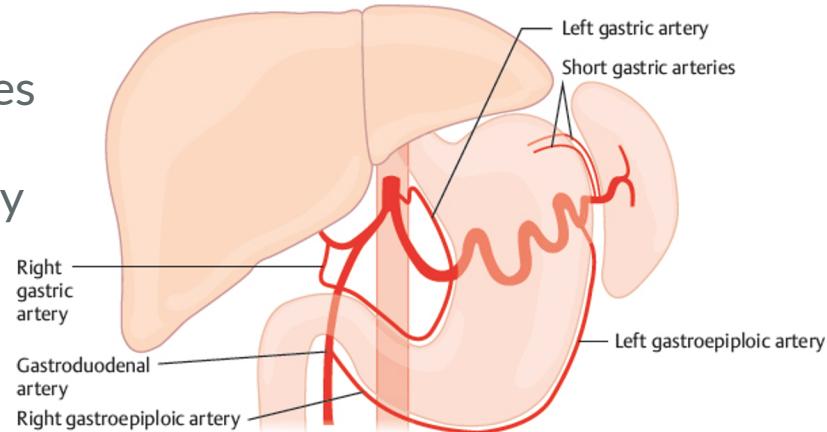
PEPTIC ULCERS- Complications

Bleeding: most common complication. Most common cause of upper GI bleeds acutely, can also cause anaemia chronically

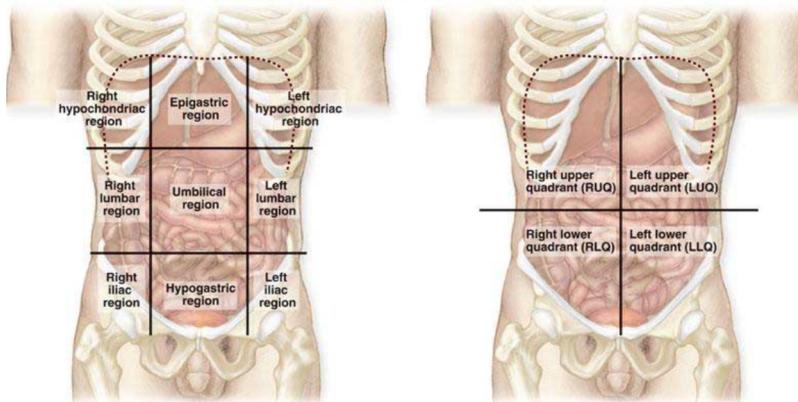
Perforation: Posterior duodenum can erode into the **gastroduodenal artery**, causing major haemorrhage. Enteric pathogens can enter peritoneum and cause inflammation and infection (peritonitis)

Gastric outlet obstruction: fibrosis and strictures

Malignancy: gastric ulcers much more commonly progress to cancer than duodenal



DDX UPPER ABDOMINAL PAIN



RIGHT UPPER	EPIGASTRIC	LEFT UPPER
<ul style="list-style-type: none"> • Cardiac • Lung- lower lobe pneumonia, PE • Liver- gallstones, cholecystitis, hepatitis hepatic congestion, liver abscess • Duodenal ulcer • Retrocaecal appendicitis (rare) 	<ul style="list-style-type: none"> • Cardiac- MI, angina, pericarditis • Aortic aneurysm or dissection • Mesenteric ischaemia or infarction • Gastric- oesophagitis, gastritis, peptic ulcer, oesophageal/gastric cancer • Pancreas- pancreatitis, pancreatic cyst, tumour 	<ul style="list-style-type: none"> • Cardiac • Lung- pneumonia, PE • Spleen- rupture, abscess, acute splenomegaly • Gastric



A 40 year old woman with a high BMI presents to the GP with heartburn, dysphagia and a chronic cough. She has also noticed that her breath smells persistently bad and this is causing her a lot of stress. What would be your next step?

- A. Screen for H. pylori infection
- B. Upper endoscopy
- C. 8 week PPI trial
- D. 8 week PPI trial with weight loss management

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A patient with persistent GORD undergoes an upper endoscopy to check for any complications of their disease. The results show Barrett's oesophagus near the lower oesophageal sphincter. The patient does not understand what this means, how would you describe it?

- A. A permanent change to the lining of the oesophagus, resulting from prolonged inflammation
- B. A change in the epithelium of the oesophagus from stratified squamous to columnar epithelium to protect against stomach acid
- C. A constriction of the oesophageal lining due to prolonged inflammation and fibrotic healing
- D. A dysplastic precancerous condition

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A 55 year old man presents to the GP with epigastric pain which goes away when he eats but then returns 2-3 hours afterwards. This has been going on for the last 2 months. The pain is 8/10 when it is at its worse, and he finds that over the counter antacids help the pain. What is the most likely diagnosis?

- A. Gastro-oesophageal reflux disease (GORD)
- B. Gastric ulcer
- C. Duodenal ulcer
- D. H pylori infection

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THANKS FOR LISTENING!

Good resources for this week:

- Armando on YouTube- great videos on stomach physiology
- Amboss for GORD and Peptic Ulcer DRDEACPIMP
- Passmedicine Textbook



<https://www.gofundme.com/f/lifesaving-bone-marrow-transplant>